Remarks

Further and favorable reconsideration is respectfully requested in view of the foregoing amendments and following remarks.

Thus, claim 7 has been cancelled, rendering the rejections of this claim under the second paragraph of 35 U.S.C. §112, and 35 U.S.C. §101 moot.

Claim 8 has been amended to delete reference to "preventing and/or", thus rendering the rejection of this claim under the second paragraph of 35 U.S.C. §112 moot.

In connection with the rejection of claim 8 under 35 U.S.C. §102(b) as being anticipated by Tada et al. (US '266), the Examiner states that it would be remedial to amend claim 8 to include terminology such as "in need thereof" when describing the mammal to be treated such that the claim establishes a clear nexus between the outcome and the method of treating. Amended claim 8 includes this expression "in need thereof", in view of which the rejection of claim 8 as being anticipated by Tada et al. has been rendered moot.

Amended claim 8 also is now set forth in independent form, having incorporated formula (II) from claim 3 in a reduced scope for the definitions of the variables. A minor editorial change has also been made, changing "administration" to "administering" in amending claim 8.

The patentability of the presently claimed invention over the disclosures of the references relied upon by the Examiner in rejecting claim 8 will be apparent upon consideration of the following remarks.

Thus the rejection of claim 8 under 35 U.S.C. §103(a) as being unpatentable over Tada et al. in view of Berdyshev et al. is respectfully traversed.

Berdyshev et al. disclose that CB agonist (WIN 55,212-2) induced a concentration-dependent decrease of TNF-α level in the bronchoalveolar lavage fluid (BALF). Additionally, they describe lipopolysaccharide (LPS) -induced bronchopulmonary inflammation in mice.

Szarka et al., cited in the Information Disclosure Statement submitted concurrently herewith, disclose that the intranasal instillation of lipopolysaccharide (LPS) into BALB/c mice causes acute pulmonary damage, due to neutrophil infiltration and sepsis (abstract). At page 56, right column, last paragraph, line 10, Szarka et al. disclose that this LPS i.n. instillation model may help continue the study of pulmonary edema, ARDS, sepsis, and toxic shock, with the understanding of therapeutic development or cytokine effectiveness.

Therefore, Applicant takes the position that the Berdyshev et al. reference cited by the Examiner does not relate to an inflammatory cell infiltration in the respiratory tract, a hyperirritability in the respiratory tract, a muciparous, or a bronchoconstrictive action.

Tada et al. disclose a CB2 agonist. However, as recognized by the Examiner, they do not disclose that the compound represented by the formula (II) in claim 8 is useful for treating an inflammatory cell infiltration in the respiratory tract, a hyperirritability in the respiratory tract, a muciparous, or a bronchoconstrictive action.

For these reasons, Applicant takes the position that the presently claimed invention is clearly patentable over the applied references.

Therefore, in view of the foregoing amendments and remarks, it is submitted that each of the grounds of rejection set forth by the Examiner has been overcome, and that the application is in condition for allowance. Such allowance is solicited.

Respectfully submitted,

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